

**COLCHICINE IN ADDITION TO STANDARD HEART FAILURE THERAPY
IN PATIENTS WITH NEW-ONSET INFLAMMATORY DILATED
CARDIOMYOPATHY**

M.M. Rakhmatov, T.A. Abdullaev, Kh.A. Gulomov.

*Republican Specialized Scientific and Practical Medical Center of Cardiology, 4 Osiyo
str., Tashkent, 100052, Uzbekistan*

Inflammation, particularly NLRP3 inflammasome activation, is increasingly recognized as one of the key driving mechanisms of myocardial dysfunction in dilated cardiomyopathy (DCM). Colchicine inhibits inflammasome assembly and has already demonstrated cardiovascular benefits in ischemic heart disease (COLCOT, LoDoCo2); however, direct data regarding DCM remain limited to a few small-scale studies. The aim of this study was to evaluate the impact of adding colchicine at a dose of 0.5 mg/day to standard chronic heart failure (CHF) therapy on echocardiographic parameters, functional status, and inflammatory/neurohormonal biomarkers in patients with new-onset DCM of a presumed inflammatory etiology.

The study included patients with new-onset DCM (symptom duration < 6 months, LVEF < 40%, NYHA class III–IV CHF, ESC 2023 criteria, presumed inflammatory phenotype) who received either standard CHF therapy combined with colchicine 0.5 mg/day (n = 23) or standard CHF therapy alone (n = 19). Baseline characteristics were comparable between the groups. Echocardiography, the 6-minute walk test (6MWT), the Clinical Severity Scale (CSS), and inflammatory/neurohormonal markers (CRP, IL-6, NT-proBNP) were assessed at baseline and after 6 months. Intra-group comparisons were performed using the Wilcoxon signed-rank test.

In the colchicine group, LVEF increased by 61.0% (from $25.3 \pm 5.6\%$ to $40.3 \pm 12.8\%$, $p = 0.038$). LVEDD and LVESD decreased by 9.9% (from 65.9 ± 6.5 mm to 59.1 ± 4.9 mm, $p = 0.015$) and 15.2% (from 56.1 ± 5.8 mm to 47.5 ± 7.6 mm, $p = 0.021$), respectively, while LVMI declined by 20.9% (from 127.8 ± 28.1 g/m² to 100.3 ± 17.4 g/m², $p = 0.017$). Concurrently, functional status improved: 6MWT distance increased by 72 m (+28.8%, $p = 0.028$), CSS score decreased by 34.1% ($p = 0.016$), and resting heart rate dropped by 16.3% ($p = 0.009$). Inflammatory markers and natriuretic peptide levels were markedly reduced: CRP by 44.5% ($p = 0.019$), IL-6 by 38.3% ($p = 0.024$), and NT-proBNP by 44.0% ($p = 0.012$). The control group receiving standard therapy alone demonstrated trends in the same direction across all parameters, but almost all changes failed to reach statistical significance (LVEF +6.8%, $p = 0.286$; LVEDD -1.2%, $p = 0.878$; LVESD -2.6%, $p = 0.594$; LVMI -7.2%, $p = 0.074$; 6MWT +11.8%, $p = 0.184$; CSS -13.9%, $p = 0.156$; HR -9.6%, $p = 0.048$; CRP -14.8%, $p = 0.238$; IL-6 -8.1%, $p = 0.521$; NT-proBNP -14.5%, $p = 0.378$).

The addition of low-dose colchicine to standard CHF therapy in patients with new-onset inflammatory phenotype DCM was associated with significant improvement in systolic function, reverse remodeling, functional capacity, and a pronounced reduction in inflammatory and neurohormonal markers. Given the non-randomized design and small sample size, these results should be viewed primarily as hypothesis-generating rather than a reason to change clinical practice; nevertheless, they provide a strong rationale for investigating colchicine in larger randomized trials for inflammatory DCM.